L Number	Hits	Search Text	DB	Time stamp
1	96076	ataxia-telangiectasia or ataxia adj telengiectasia or ATM	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:42
2	564	ataxia-telangiectasia or ataxia adj telengiectasia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:43
3	91	(ataxia-telangiectasia or ataxia adj telengiectasia) and ATM	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:11
4	22	(ataxia-telangiectasia or ataxia adj telengiectasia) and ATM same (deficient or deleted) same cell	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:46
5	5	ATM adj (deficient or deleted) adj cell	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:25
6	10	ATM adj (deficient or deleted) same cell	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:58
8	460	(ataxia-telangiectasia or ataxia adj telengiectasia) and virus	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:59
9	10	(ATM adj (deficient or deleted) same cell) and virus	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:59
10	289	ATM same (cloning or clone or subclone or vector)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:13
11	20	(ATM same (cloning or clone or subclone or vector)) and vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:25
12	52	Mec1	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:24
13	25	Mec1 and ATM	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:24
14	63	ATM and (deficient or deleted) adj cell	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:32

15	13	(ATM and (deficient or deleted) adj cell) and vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:25
16	4	Mecl and (deficient or deleted) adj cell	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:37
17	3	(Mecl and (deficient or deleted) adj cell) and atm	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 10:37
-	358	ataxia-telangiectasia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2003/07/30 09:41
-	147999	(viral or virus) same mammalian	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:22
-	215	ataxia-telangiectasia and ((viral or virus) same mammalian)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:22
-	214	(ataxia-telangiectasia and ((viral or virus) same mammalian)) and expression	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:23
-	1	(ataxia-telangiectasia and ((viral or virus) same mammalian)) and expresion	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:23
-	44	ataxia-telangiectasia same expression	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:25
-	3	(ataxia-telangiectasia same expression) and ((viral or virus) same mammalian)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:23
-	61	ataxia-telangiectasia same protein	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:25
-	45	(ataxia-telangiectasia same protein) and (virus or adenovirus or viral adj vector)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:33
-	8	ataxia-telangiectasia same (virus or adenovirus or viral adj vector)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:27
-	48	ataxia-telangiectasia same (expression or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:27

-	2075558	ataxia-telangiectasia adj10\ (expression or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:29
-	0	ataxia-telangiectasia adj10 (expression or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:29
-	2	ataxia-telangiectasia adj20 (expression or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:29
-	4	(ataxia-telangiectasia same protein) same (virus or adenovirus or viral adj vector)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 15:34
-	102	ataxia-telangiectasia same polypeptide	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:09
-	90	(ataxia-telangiectasia same polypeptide) and production	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:09
-	0	((ataxia-telangiectasia same polypeptide) and production) and Barlow	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:10
-	31	"0112647"	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:10
-	0	"0112647" and barlow	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:10
-	8637	Barlow, Carolee	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:10
-	8838	Callahan	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:11
-	8	Callahan and Barlow	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/11 16:11
-	206	ataxia-telangiectasia and vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:21
-	262911	method adj2 producing	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:17

-	139	(method adj2 producing) same vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:15
-	0	((method adj2 producing) same vaccinia) and (ataxia-telangiectasia and vaccinia)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:15
-	188	(method adj2 producing) and (ataxia-telangiectasia and vaccinia)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:15
-	188	((method adj2 producing) and (ataxia-telangiectasia and vaccinia)) and virus	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:15
-	0	ataxia-telangiectasia same (method adj2 producing)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:21
-	0	(method adj2 producing) same vaciinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:16
-	138	(method adj2 producing) same vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:17
-	240728	method adj1 producing	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:18
-	137	(method adj1 producing) same vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:21
-	16	(method adj1 producing) adj10 vaccinia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:51
-	147	(method adj1 producing) same (vaccinia or poxvirus)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:21
-	0	ataxia-telangiectasia and ((method adj1 producing) same (vaccinia or poxvirus))	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:21
-	0	ataxia-telangiectasia same (method adj2 producing)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:22
-	209	ataxia-telangiectasia and (method adj2 producing)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:22

-	188	(ataxia-telangiectasia and (method adj2 producing)) and (vaccinia or poxvirus)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/18 12:22
-	1	pSCAT	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:34
-	10	barlow and ataxia adj telangiectasia	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:52
-	50	ataxia adj telangiectasia adj gene	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:54
-	35	(ataxia adj telangiectasia adj gene) and (virus or viral or adenovirus or retrovirus)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:54
-	0	(ataxia adj telangiectasia adj gene) same (virus or viral or adenovirus or retrovirus)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:55
-	0	ataxia adj telangiectasia adj gene same expression	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:54
-	67	ATM same (virus or viral or adenovirus or retrovirus)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:56
-	28	(ATM same (virus or viral or adenovirus or retrovirus)) and tumor	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:56
-	2	ATM adj expression	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:56
-	67	ATM same (virus or viral or adenovirus or retrovirus or vaccinia)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:59
-	43	(ATM same (virus or viral or adenovirus or retrovirus or vaccinia)) and (express or expression or produce or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:58
-	32181	ATM and (express or expression or produce or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:59
-	5042	ATM same(express or expression or produce or production	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 18:03

-	134	(ATM same(express or expression or produce or production)) and (virus or viral or adenovirus or retrovirus or vaccinia)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:59
-	72	(ATM same(express or expression or produce or production)) and (adenovirus or retrovirus or vaccinia)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 17:59
-	53	(((ATM same(express or expression or produce or production)) and (adenovirus or retrovirus or vaccinia)) and kinase	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 18:00
-	13	((((ATM same(express or expression or produce or production)) and (adenovirus or retrovirus or vaccinia)) and kinase) and (DNA adj repair)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 18:01
-	3	ATM adj kinase same (express or expression or produce or production)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 18:03
-	2	(ATM adj kinase same (express or expression or produce or production)) and virus	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM_TDB	2002/12/19 18:03

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NEWS 19  May 19   Simultaneous left and right truncation added to WSCA
NEWS 20  May 19   PAIRA enhanced with new search field, simultaneous left and
                  right truncation
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NEWS 22  Jun 16   PAPOL enhanced with additional data
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NEWS 26  Jul 21   Identification of STN records implemented
NEWS 27  Jul 21   Polymer class term count added to REGISTRY
NEWS 28  Jul 22   INPADOC: Basic index (/BI) enhanced; Simultaneous Left and
                  Right Truncation available

NEWS EXPRESS  April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT
              MACINTOSH VERSION IS V6.0b ENG  AND V6.00b CFC,
              AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2014
NEWS HOURS    STN Operating Hours Plus Help Desk Availability
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FILE 'HOME' ENTERED AT 12:12:24 ON 21 APR 2014

=> file caplus
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=> s ATH 4, deficient  A  well
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```

[illegible]
PUBMED ABS: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB [illegible] deficient cells. ATM in ATM deficient cells [illegible] response [illegible] ATM [illegible] We expressed the ATM kinase in AT cells to test the DNA damage responsibility [illegible], whether the p74 kinase activity [illegible] ATM [illegible] The data suggest a role for the ATM kinase in maintaining the intracellular concentration of tyrosine-phosphorylated [illegible] regulate p74 expression. Thus, IRF-1 may be an early downstream signaling pathway, a new ATM-dependent signaling pathway in normal cells, and an ATM-independent pathway in DNA damaged cells.

L4 ANSWER [illegible] MEDLINE [illegible]
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible]
TITLE: ATM is activated in response to IR-mimetic, NF-kappa B-activating and DNA damage-unrelated stimuli.
AUTHOR: Aikawa Akari W; Pro Wanudy; Chaudhary Kaleem; Kamagata K; Duran Pedro I
CORP SOURCE: Department of Biochemistry and Molecular Biology and the Graduate Center, [illegible] Cancer Center, [illegible] Health Sciences Center, New Orleans, Louisiana [illegible], USA.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2002 Oct 11)
[illegible] 41 [illegible].
Journal code: [illegible]. ISSN: [illegible].
PUB. TYPE: Entered STN:
DOCUMENT TYPE: Journal; Article; JOURNAL ARTICLE
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB p74 plays an important role in response to ionizing radiation by regulating cell cycle progression and triggering apoptosis. These activities are controlled, in part, by the phosphorylation of p74 by the protein kinase ATM. Recent evidence indicates that the mutations that DNA alkylating agents thereby, [illegible] nitrogen mustards [illegible] trigger p74 activation and phosphorylation of p74; however, the mechanism [illegible] responsible for this are unknown. We reported that in MMNG-treated normal human fibroblasts, up-regulation and phosphorylation of p74 was sensitive to the ATM kinase inhibitor wortmannin. ATM-deficient human lymphoblasts exhibited a delay in p74 up-regulation, [illegible] a role for ATM in triggering the MMNG-induced response. Likewise, a mismatch repair MMR deficient colorectal tumor line failed to show rapid p74 regulation. However, unlike ATM deficient cells, these MMR-deficient cells displayed rapid phosphorylation of the p74 residue serine [illegible] after MMNG. In vitro kinase assays indicate that ATM is rapidly activated in both normal and MMR deficient cells in response to MMNG. Using a number of complementary approaches, we failed to observe MMNG-induced apoptosis in normal human fibroblasts, suggesting that apoptosis-inducing DNA strand breaks are not required for the activation of ATM in response to MMNG. These assays indicated that strand breaks accumulated, and p74 [illegible] phosphorylation occurred quite rapidly, within 45 min, after MMNG treatment, suggesting that DNA strand breaks during the repair process activate ATM. These findings indicate that ATM activation [illegible] the structural alterations induced by some and potentially play a role in triggering DNA damage [illegible].

L4 ANSWER [illegible] MEDLINE [illegible]
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible]
TITLE: DNA-PK and ATM are required for telomere [illegible] processing.
AUTHOR: Hande M Prakash; Samper Elisa; Lansdorp P; Blasco Maria A

AUTHOR(S): Department of Experimental Radiology, The University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, Texas 77030, USA.
JOURNAL ARTICLE:
(PUBLICATION TYPE)
SOURCE: MEDLINE
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020222
Last Updated on STN: 20030117
Entered Medline: 20020419
AB Ionizing radiation (IR) is known to activate multiple cell cycle checkpoints that are thought to enhance the ability of cells to repair DNA damage. Protein phosphatase 2A (PP2A) has been implicated in IR-induced activation of checkpoints; therefore, tumor cells were exposed to an activating dose of IR or sham treatment and control and nuclear extracts were analyzed for PP2A by Mono Q anion exchange chromatography and microcystin affinity chromatography. PP2A activity in eukaryotic cells is a heterotrimer consisting of a 65-kDa scaffolding subunit (A), plus a 36-kDa regulatory subunit (B) and an ABC heterotrimeric, containing one of a variety of regulatory B and subunits. Here we show that IR promotes translocation and retention in the nucleus of nuclear ABC heterotrimer without affecting the ABC heterotrimer or Ab heterodimer. To enable molecular examination of **ATM deficient cells** the amount of nuclear PP2A heterotrimer retained in heterotrimer was not reduced by radiation, but the radiation response was restored by transformation of these cells with plasmids encoding ATM. Furthermore, an inhibitor of known protein serine/threonine phosphatases, okadaic acid, also prevented the IR-induced retention in nuclear PP2A heterotrimer. The changes in nuclear PP2A activities without a significant difference in the methylesterification of the C terminus, which is known to influence interaction with B subunits. We conclude a novel ATM dependent mechanism is regulating nuclear retention of BPP2A with nuclear PP2A in

24 ANSWER 2 OF 10 MEDLINE on STN
ACCESSION NUMBER: 2002211919 MEDLINE
DOCUMENT NUMBER: 20542401 PubMed ID: 11924140
TITLE: ATM dependent silencing of IRF-1 repressor-mediated cell proliferation in response to ionizing radiation.
AUTHOR: Sun Chang Y; Hobson Gordon David L; Daniel James M
CORPORATE SOURCE: Department of Radiation Oncology, University of Virginia Health Sciences System, Charlottesville, Virginia 22908, USA.
JOURNAL NUMBER: NAR26622 (JOURNAL)
JOURNAL: JOURNAL OF BIOLOGICAL CHEMISTRY, (2002 Feb 15)
Vol. 277, No. 7, pp. 4949-54.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020222
Last Updated on STN: 20030117
Entered Medline: 20020419
AB Ionizing radiation (IR) is known to activate multiple cell cycle checkpoints that are thought to enhance the ability of cells to repair DNA damage. Protein phosphatase 2A (PP2A) has been implicated in IR-induced activation of checkpoints; therefore, tumor cells were exposed to an activating dose of IR or sham treatment and control, and nuclear extracts were analyzed for PP2A by Mono Q anion exchange chromatography and microcystin affinity chromatography. PP2A activity in eukaryotic cells is a heterotrimer consisting of a 65-kDa scaffolding subunit (A), plus a 36-kDa regulatory subunit (B) and an ABC heterotrimeric, containing one of a variety of regulatory B and subunits. Here we show that IR promotes a transient and reversible retention in the nucleus of nuclear ABC heterotrimer without affecting the ABC heterotrimer or Ab heterodimer. To enable molecular examination of **ATM deficient cells** the amount of nuclear PP2A heterotrimer retained in heterotrimer was not reduced by radiation, but the radiation response was restored by transformation of these cells with plasmids encoding ATM. Furthermore, an inhibitor of known protein serine/threonine phosphatases, okadaic acid, also prevented the IR-induced retention in nuclear PP2A heterotrimer. The changes in nuclear PP2A activities without a significant difference in the methylesterification of the C terminus, which is known to influence interaction with B subunits. We conclude a novel ATM dependent mechanism is regulating nuclear retention of BPP2A with nuclear PP2A in

response to IR.

L4 ANSWER 17 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2001[illegible] MEDLINE
DOCUMENT NUMBER: 21[illegible] PubMed ID: 11[illegible]
TITLE: ATM-dependent phosphorylation of human Rad9 is required for ionizing radiation-induced checkpoint activation.
AUTHOR: Chen M J; Lin Y T; Lieberman H B; Chen G; Lee E Y
CORPORATE SOURCE: Department of Molecular Medicine/Institute of Biotechnology, The University of Texas Health Science Center, San Antonio, Texas 78245-3207, USA.
CONTRACT NUMBER: CA81 [illegible] (NCI)
[illegible] (NIGMS)
[illegible] (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 May 11) 276 (19) 16580-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010618
Last Updated on STN: 2001[illegible]
Entered Medline: 20010614

AB ATM (ataxia telangiectasia mutated) is a Ser/Thr kinase involved in cell cycle checkpoints and DNA repair. Human Rad9 (hRad9) is the homologue of Schizosaccharomyces pombe Rad9 protein that plays a critical role in cell cycle checkpoint control. To examine the potential signaling pathway linking ATM and hRad9, we investigated the modification of hRad9 in response to DNA damage. Here we show that hRad9 protein is constitutively phosphorylated in undamaged cells and undergoes hyperphosphorylation upon treatment with ionizing radiation (IR), ultraviolet light (UV), and hydroxyurea (HU). Interestingly, hyperphosphorylation of hRad9 induced by IR is dependent on ATM. Ser(272) of hRad9 is phosphorylated directly by ATM in vitro. Furthermore, hRad9 is phosphorylated on Ser(272) in response to IR in vivo, and this modification is delayed in **ATM deficient cells**. Expression of hRad9 S272A mutant protein in human lung fibroblast VA13 cells disturbs IR-induced G(1)/S checkpoint activation and increases cellular sensitivity to IR. Together, our results suggest that the ATM-mediated phosphorylation of hRad9 is required for IR-induced checkpoint activation.

L4 ANSWER 18 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2001309[illegible]4 MEDLINE
DOCUMENT NUMBER: 21225[illegible] PubMed ID: 11331310
TITLE: Tumor suppressor p53 binding protein 1 (53BP1) is involved in DNA damage-signaling pathways.
COMMENT: Erratum in: J Cell Biol 2001 Jul 23;154(2):469
AUTHOR: Rappold I; Iwabuchi K; Date T; Chen J
CORPORATE SOURCE: Division of Oncology Research, Mayo Clinic, Rochester, Minnesota 55905, USA.
SOURCE: JOURNAL OF CELL BIOLOGY, (2001 Apr 30) 153 (3) 613-20.
Journal code: [illegible] ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 2001[illegible]
Last Updated on STN: 2001[illegible]
Entered Medline: 2001[illegible]

AB The tumor suppressor p53 binding protein 1 (53BP1) binds to the DNA-binding domain of p53 and enhances p53-mediated transcriptional activation. 53BP1 contains two breast cancer susceptibility gene 1 COOH terminus (BRCT) motifs, which are present in several proteins involved in DNA repair and/or DNA damage-signaling pathways. Thus, we investigated the potential role of 53BP1 in DNA damage-signaling pathways. Here, we report that 53BP1 becomes hyperphosphorylated and forms discrete nuclear foci in response to DNA damage. These foci colocalize at all time points with phosphorylated H2AX (gamma-H2AX), which has been previously demonstrated to localize at sites of DNA strand breaks. 53BP1 foci formation is not restricted to gamma-radiation but is also detected in response to UV radiation as well as hydroxyurea, camptothecin, etoposide, and methylmethanesulfonate treatment. Several observations suggest that 53BP1 is regulated by ataxia telangiectasia mutated (ATM) after DNA damage. First, **ATM deficient cells** show no 53BP1 hyperphosphorylation and reduced 53BP1 foci formation in response to gamma-radiation compared with cells expressing wild-type ATM. Second,

...with normal treatment of fully ionizing irradiation or with...
...agent that activates the ATM kinase. These results suggest...
...that ATM is an ATM substrate that is involved early in the DNA...
...damage response pathway in T-cell...

L4 ANSWER 4 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2...
DOCUMENT NUMBER: ...
TITLE: The p...an ATM dependent manner.
AUTHORS: ...
CORPORATE SOURCE: ...
SOURCE: ...
PUB. COUNTRY: ...
DOCUMENT TYPE: ...
LANGUAGE: ...
FILE SEGMENT: ...
ENTRY MONTH: ...
ENTRY DATE: ...

AB ...ataxia-telangiectasia mutated (ATM) kinase is required for...
...cell cycle checkpoint activation following exposure to ionizing...
...radiation and other genotoxic agents. ATM is known to...
...phosphorylate p53 at serine 15, but the substrates for ATM that...
...mediate activation of the G2 checkpoint have not been...
...identified. We show that ATM is required for the...
...phosphorylation of p53 at serine 15, but not for the...
...phosphorylation of Chk2 at threonine 68, following exposure of...
...cells to gamma-irradiation. Phosphorylation and...
...accumulation of p53 and phosphorylation of Chk2 were also observed in **ATM deficient cells**. In contrast, the...
...gamma-irradiation-induced phosphorylation of p53 and Chk2 in ATM-positive and **ATM deficient cells**. In addition, genistein-treated **ATM deficient cells** were significantly more susceptible to genistein-induced killing than were ATM positive cells. Together our data suggest that ATM is required for activation of a DNA damage-induced pathway that activates p53 and Chk2 in response to genistein.

L4 ANSWER 5 OF 12 MEDLINE on STN
ACCESSION NUMBER: ...
DOCUMENT NUMBER: ...
TITLE: Synthetic lethality between mutation in Atm and DNA-PK cs during murine embryogenesis.
AUTHORS: ...
CORPORATE SOURCE: ...
SOURCE: ...
PUB. COUNTRY: ...
DOCUMENT TYPE: ...
LANGUAGE: ...
FILE SEGMENT: ...
ENTRY MONTH: ...
ENTRY DATE: ...

AB The gene product mutated in ataxia telangiectasia, ATM, is a ubiquitously expressed ATI-like protein kinase that is a key mediator of the cellular response to DNA damage [1]. **ATM deficient cells** are radiosensitive and show impaired cell cycle arrest and in vitro abnormalities in response to a limited range of ... ATM is a member of the phosphatidylinositol 3'-kinase (PI3K)-related protein kinase (PIKK) family, which includes the catalytic subunit of DNA dependent protein kinase (DNA-PKcs) and ATR [2]. DNA-PKcs is a PIKK protein kinase that is required for proper end-joining of DNA double-strand breaks [3]. Like wild-type mice, mice lacking DNA-PKcs are viable and radiosensitive [4-6]. To determine if Atm and DNA-PKcs show genetic interaction, we attempted to generate mice deficient in both genes [7,8]. However, no viable Atm-/-; ... pups were recovered from an intercross of Atm+/- ... intercross mice. Developmental arrest of Atm-/-; ... embryos occurred

[illegible] ATM and DNA-PK, a novel potential therapeutic target for [illegible] DNA damage [illegible]. These results suggest a potential interplay between ATM and DNA-PK and indicate that ATM and DNA-PK have complementary roles that are essential for [illegible] development.

L4 ANSWER 11 OF 12 MEDLINE on STN
ACCESSION NUMBER: [illegible] MEDLINE
ENTRY NUMBER: [illegible] PubMed ID: [illegible]

TITLE: Partial Attenuation of Disease Phenotypes in Ataxia-Telangiectasia Mutated Knockout Mice Heterozygous for a Common Hypomorphic Allele.

AUTHOR: Gumbs M.R.A.; Kysela B.; Li J.; Jeggo P.A.; Jones N.J.
CORPORATE SOURCE: Medical Research Council Department of Medical Genetics, University of Manchester, Manchester, United Kingdom.

SOURCE: Molecular and Cellular Biology, (2000 Nov-Dec) [illegible]
[illegible].
Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB We previously reported that heterozygosity for a prevalent [illegible] mutation in Mediterranean populations, and that the [illegible] mutation [illegible] cell cycle arrest, and that the [illegible] mutation [illegible] cell cycle arrest [illegible] with type [illegible]. These observations [illegible] that we now report that three different ATM mutations also attenuate cell cycle arrest [illegible] with type ATM when heterozygous in either Mediterranean or an ATM deficient cell line. We determined [illegible] differences in the degree of [illegible] these ATM mutations exert. ATM [illegible] retained [illegible] in [illegible] the most [illegible] and [illegible] with the ATM [illegible] mutation [illegible] the least [illegible], with the ATM [illegible] mutation [illegible] the [illegible] [illegible] compared with type ATM. The degree [illegible] within the different mutations correlated [illegible] cycle progression [illegible] with the age of ATM [illegible] by the mutation in question. [illegible] analysis of protein extracts from prevalent [illegible] heterozygous cells indicated that the cell cycle regulated cytoplasm [illegible] pool of [illegible] is dramatically [illegible], whereas the [illegible] [illegible] pool remains essentially unaffected. We discuss the [illegible] of these findings in relation [illegible] to cell cycle arrest, apoptosis and AD.

L4 ANSWER 11 OF 12 MEDLINE on STN
ACCESSION NUMBER: [illegible] MEDLINE
ENTRY NUMBER: [illegible] PubMed ID: [illegible]

TITLE Regulation of DNA-dependent protein kinase activity by [illegible] variation within a cell line lacking an ATM-dependent [illegible].

AUTHOR: Chaudhary J.; Brown P.D.; Alexander A.W.; Pearson J.; Yu B.; Pandita T.K.; Yalowich J.; Marcelli P.B.; Baskaran R.
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry and the Department of Pharmacology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania [illegible], USA.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Sep 29)
[illegible].
Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB [illegible] treatment results in activation of the heterotrimeric [illegible] DNA-dependent protein kinase (DNA-PK) and its phosphorylation and [illegible] potentially activate ATM kinase activity in response to IR exposure. To unravel the role of ATM and DNA-PK in the activation of ATM, we analyzed ATM, ATM, and DNA-PK activity in ATM and DNA-PK cell lines after irradiation. [illegible] results show that despite the presence of [illegible] levels of DNA-PK kinase activity, ATM fails to be activated after IR exposure in ATM deficient cells. Conversely, normal activation of the ATM and ATM [illegible] in DNA-PK deficient cells, indicating that ATM is

[illegible] DNA-PK is required for activation of the ATM protein kinase following gamma irradiation. These results suggest that ATM activity in all phases of the cell cycle. These results are consistent with ATM being primarily responsible for activation of the p53 protein following DNA damage in G1 cell cycle independent of DNA-PK activity. In contrast, the ATM protein kinase is not required for activation of p53 following gamma irradiation in G1 phase cells. While [illegible] these results suggest a convergence of the ATM and DNA-PK pathways on the cellular response to DNA damaging agents.

1/ ANSWER 12 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2000[illegible] MEDLINE
DOCUMENT NUMBER: 2000[illegible] PubMed ID: [illegible]
TITLE: Ionizing radiation induces [illegible] kinase activity, [illegible] phosphorylation of [illegible] protein after gamma irradiation to different doses.
AUTHOR: Khanna K.K.; Watanabe M.; Taya Y.; Shiloh Y.
CORPORATE SOURCE: Department of Clinical Pathology, Showa University, School of Medicine, Tokyo, Japan. KKK@intmed.med.showa-u.ac.jp
JOURNAL: Biochemical and Biophysical Research Communications, (2000 Apr 17) 1[illegible]
[illegible]
Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]
AB The [illegible] kinase is the product of the gene responsible for the [illegible] DNA damage is required for efficient p21 expression by demonstrating that enhanced p21 mRNA expression induced by DNA damage results in increased p21 protein, but enhanced p21 mRNA without DNA damage does not. In addition, we demonstrated that DNA damage increased the phosphorylation of p21. In this study, we analyze the link between p21 stabilization and DNA damage. Enhanced p21 protein expression in ML-1 cells resulting from 10 Gy gamma irradiation was diminished by Wortmannin or LY294002 pretreatment of cells. However, the levels of p21 mRNA were not affected by inhibitor pretreatment. Wortmannin or LY294002 pretreatment reduced p21 expression after gamma-irradiation to a lesser degree than that of p21 [illegible]. In addition, we examined the involvement of DNA-PK, whose activity is inhibited by Wortmannin or LY294002, in p21 stabilization using the SCID fibroblast cell line and a DNA-PK targeting ML-1 cell line. Accumulation of p21 protein by gamma irradiation was similar to that in DNA-PK intact cells and was reduced by Wortmannin or LY294002 pretreatment. Involvement of another DNA damage detecting enzyme, the ATM gene product, whose activity is also inhibited by Wortmannin or LY294002, was evaluated. ATM deficient cells induced p21 after gamma irradiation, gamma irradiation-induced p21 protein was diminished by pretreatment of cells with Wortmannin or LY294002. We conclude that the p21 stabilization mechanism functions after gamma irradiation, was sensitive to Wortmannin or LY294002, and required neither DNA-PK nor ATM gene product for activity.

14/ ANSWER 14 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2[illegible] MEDLINE
DOCUMENT NUMBER: 2[illegible] PubMed ID: [illegible]
TITLE: ATM: a mediator of multiple responses to genotoxic stress.
AUTHOR: Rotman G.; Shiloh Y.
CORPORATE SOURCE: Department of Human Genetics and Molecular Medicine, Sackler Faculty of Medicine, Tel Aviv University, Ramat Aviv 69978, Israel.
JOURNAL: [illegible] (1999 Nov 1) 1[illegible]
Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: England
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]
AB The ATM protein kinase is the product of the gene responsible for the

[illegible] ATM **deficient cells** [illegible]

L4 ANSWER 14 OF 19 MEDLINE on STN
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible] PubMed ID: [illegible]
TITLE: [illegible]
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUB. TYPE: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]
AB [illegible] **Atm deficient cells.** [illegible]

L4 ANSWER 15 OF 19 MEDLINE on STN
ACCESSION NUMBER: 1999413496 MEDLINE
DOCUMENT NUMBER: 99413496 PubMed ID: 10468496
TITLE: Interaction of ATM and ATP during inhibition by the
[illegible]
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUB. TYPE: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]
AB [illegible]

with the kinetics of multiple DNA damage-induced cell cycle checkpoints. The observation that wortmannin, but not caffeine, abrogates radiation-induced G2 arrest in ATM-deficient cells suggests that caffeine may inhibit the radiation-induced G2 checkpoint in an ATM-deficient cell through an ATM-independent pathway in DNA-damaged cells. We now show that caffeine inhibits the catalytic activity of the ATM and the related kinase, ATM and Rad3-related (ATR), at concentrations similar to those that inhibit radiation-induced G2 checkpoint, like ATM deficient cells. Caffeine treated A-T cells lose the ability to arrest progression into mitosis, and irradiated cells exhibit premature chromosome condensation. Similar to caffeine, overexpression of kinase-dead ATM and ATR mutants also inhibits radiation-induced G2 checkpoint, whereas overexpression of wild-type ATM and ATR does not. Caffeine, like the dominant-negative mutants, also inhibits DNA damage-induced activation of the cyclin B1-associated cdc2 kinase. Finally, caffeine, like the dominant-negative mutants of ATM and ATR, but not wortmannin, sensitizes human tumor cells to ionizing radiation. These data suggest that the radiosensitization effect of caffeine is related to inhibition of the radiation-induced kinase activities of ATM and ATR and that both proteins are relevant targets in the development of novel antitumor agents.

24. ANSWER 17 OF 19 MEDLINE on STN
ACCESSION NUMBER: 1998414417 MEDLINE
DOCUMENT NUMBER: 98414417 PubMed ID: 9722607
TITLE: Inhibition of phosphatidylinositol 3-kinase related kinases by the fungal metabolite wortmannin.
AUTHOR: Sarkaria J N; Tibbetts R S; Busby E C; Kennedy A P; Hill D E; Abraham R T
CORPORATE SOURCE: Department of Pharmacology, Mayo Clinic, Rochester, Minnesota 55905, USA.
SOURCE: CANCER RESEARCH, (1998 Oct 1) 58 (19) 4375-82.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19981016
Last Updated on STN: 20120402
Entered Medline: 19981104
AB Members of the phosphatidylinositol 3-kinase related kinase (PIKK) family function in both cell cycle progression and DNA damage-induced cell cycle checkpoints. The fungal metabolite, wortmannin, is an effective radiosensitizer that irreversibly inhibits certain members of the PIKK family. Based on their roles in DNA damage responses, several PIKKs, DNA dependent protein kinase (DNA-PK), ataxia telangiectasia mutated (ATM) and the ataxia- and Rad3-related protein (ATR), are potential targets for the radiosensitization effect of wortmannin. In this report, we demonstrate that wortmannin is a relatively potent inhibitor of DNA-PK (IC50, 16 nM) and ATM (IC50, 150 nM) autokinases, whereas ATR activity is significantly less sensitive to this drug (IC50, 1.8 microM). In intact A549 lung adenocarcinoma cells, wortmannin inhibited both DNA-PK and ATM at concentrations that correlated closely with those required for radiosensitization. Furthermore, pretreatment of A549 cells with wortmannin resulted in radioresistant DNA synthesis, a characteristic abnormality of ATM deficient cells. These results identify wortmannin as an inhibitor of both ATM activity and without that ATM and DNA-PK are relevant targets for the radiosensitization effect of this drug in tumor cells.

25. ANSWER 18 OF 19 MEDLINE on STN
ACCESSION NUMBER: 1998161196 MEDLINE
DOCUMENT NUMBER: 98161196 PubMed ID: 9496465
TITLE: Atm selectively regulates distinct p53-dependent cell-cycle checkpoint and p53-independent pathways.
AUTHOR: Barlow C; Brown K D; Deng C X; Tagle D A; Wynshaw-Boris A
CORPORATE SOURCE: Laboratory of Genetic Disease Research, National Institute of Diabetes, Digestive and Kidney Diseases, Bethesda, Maryland 20892, USA.
SOURCE: DNA REPAIR, (1997 Dec) 1 (12) 871-83.
Journal code: 101170634. ISSN: 1568-7864.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812

[illegible] DATE: Entered: [illegible]
Last updated on [illegible]
Entered Medline: [illegible]

AB ATM is part of a pathway that responds to DNA damage in the nucleus. Loss of ATM activity results in the [illegible] **Atm deficient cell** lines, which are also defective in [illegible] lines, after IR, progress through the cell cycle with [illegible] However, the mechanism by which ATM differentially activates downstream pathways is unknown. To determine the relationship between ATM and p53, we examined cell cycle and apoptotic responses in ATM, p53 single, and ATM/p53 double deficient mice after IR in the whole animal. As expected, p53 function levels were not influenced by IR in thymus [illegible] ATM deficient mice. IR induced cell cycle checkpoint [illegible] defective, and this defect in p53 was attributed to dysfunction of ATM and not tissue type. However, IR induced apoptosis was [illegible] were completely normal, in that it was not related to p53. IR induced thymic apoptosis was suppressed in ATM p53 double mutants, demonstrating p53 dependence and ATM independence. Thus, ATM deficiency results in [illegible] p53 dependently by IR, but only delays thymic [illegible] p53-dependent functions. These results suggest a model in which ATM has a direct effect on which an ATM deficiency activates p53 to initiate apoptosis downstream pathways, or [illegible] downstream cell cycle and apoptotic responses.

AU AUTHOR [illegible] MEDLINE [illegible]
ACCESSION NUMBER: [illegible] MEDLINE
[illegible] NUMBER: [illegible] PubMed ID: [illegible]
TITLE: The [illegible] gene is a [illegible] that [illegible] the human [illegible] gene.
AUTHORS: Hall P.; Pandita R.; Deveraux Q.; Miller P.; Bryant P.; Hawley R.
CORPORATE AUTHOR: Department of Genetics, University of [illegible], Dallas, [illegible], USA.
CITED: Cell, (1995 Sep 8) [illegible].
Journal Code: [illegible]. ISSN: [illegible].
PUB. COUNTRY United States
[illegible] TYPE: Journal; Article; JOURNAL ARTICLE
LANGUAGE English
FILE SEGMENT Priority Journals
OTHER ABS GENBANK U49928
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951106
Last updated on STN: 19951106
Entered Medline: 19951101

AB The [illegible] gene is required for DNA repair, mitotic chromosome stability, and normal levels of meiotic recombination in [illegible]. Here we show that the predicted mei-41 protein is similar in sequence to the ATM [illegible] protein from humans and to the yeast rad3 and Mec1 proteins. There is also extensive functional overlap between mei-41 and ATM. Like **ATM-deficient cells**, mei-41 cells are exquisitely sensitive to ionizing radiation and display high levels of mitotic chromosome instability. We also demonstrate that mei-41 cells, like **ATM-deficient cells**, fail to show an irradiation-induced delay in the entry into mitosis that is characteristic of normal cells. Thus, the mei-41 gene of Drosophila may be considered to be a functional homolog of the human ATM gene.

AU AUTHOR [illegible] "AIELLO COPYRIGHT 2000 ACS on STN
ACCESSION NUMBER: [illegible] "AIELLO
[illegible] NUMBER: [illegible]
TITLE: Atm deficient mice Purkinje cells show age dependent defects in calcium spike bursts and calcium currents
AUTHORS: Inoue, M.; Parker, A.; Wyatt-Haston, A.; Brown, P.; Temple, F.
CORPORATE AUTHOR: Department of Neuroscience, University of Turin, Turin, Italy
CITED: Neuroscience [illegible] 2000, [illegible] CODEN: NRSCDN; ISSN: [illegible]
PUBLISHER: Elsevier Science B.V.
[illegible] TYPE: Journal
LANGUAGE English

AB Ataxia telangiectasia in human results in [illegible] Atm deficient mice in ATM. Here [illegible] demonstrate that the Purkinje cells of Atm [illegible] ataxia telangiectasia patients in the cerebellum, mainly Purkinje cells are affected. We have generated ATM-deficient mice which display neurological abnormalities by general tests. [illegible] with an age dependency in cerebellar functions, but without overt [illegible] neurological abnormalities. Here we performed a more detailed morphological and